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VASCULAR DISEASE

DOSE DEPENDENT EFFECTS OF ATORVASTATIN ON ENDOTHELIAL FUNCTION, ARTERIAL STIFFNESS AND INFLAMMATORY STATUS IN ISCHEMIC HEART FAILURE PATIENTS

ACC Poster Contributions

Ernest N. Morial Convention Center, Hall F

Monday, April 04, 2011, 3:30 p.m.-4:45 p.m.

Session Title: Peripheral Arterial/Carotid Disease/Aortic Disease

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Background: Heart failure (HF) is characterized by increased inflammatory status and impaired endothelial function. Statins beyond its lipid lowering effect exert beneficial effect on endothelial function and inflammatory process in patients with atherosclerosis. We examined the impact, of different doses of atorvastatin treatment, on arterial wall properties and on inflammatory status in HF patients.

Methods: We studied the effect of 4 weeks administration of atorvastatin in 17 patients with ischemic HF. The study was carried out on two separate arms, one with atorvastatin 40 mg/d and one with atorvastatin 10 mg/d (randomized, double-blind, cross-over design). Measurements were carried out at baseline and at the end of each treatment period. Endothelial function was evaluated by flow mediated dilation (FMD) in the brachial artery and arterial stiffness was evaluated by Augmentation Index (Alx). Serum levels of TNF- α and BNP were measured as an inflammatory marker and as an index of heart failure status respectively.

Results: Compared to baseline, treatment with 40 mg/d of atorvastatin improved FMD ($3.48 \pm 2.89\%$ vs. $6.17 \pm 2.61\%$, $p < 0.01$) and Alx ($27.49 \pm 7.83\%$ vs. $22.83 \pm 8.71\%$, $p < 0.05$). Moreover, between the low and high dose of atorvastatin treatment there was a significant increase in FMD ($3.73 \pm 1.73\%$ vs. $5.86 \pm 2.42\%$, $p < 0.01$) and decrease in Alx ($26.45 \pm 7.86\%$ vs. $22.83 \pm 8.71\%$, $p < 0.05$). However, treatment with 10 mg/d of atorvastatin did not improved FMD ($3.76 \pm 1.67\%$ vs. $3.36 \pm 2.23\%$, $p = 0.52$) and Alx ($25.89 \pm 7.89\%$ vs. $27.13 \pm 7.83\%$, $p = 0.317$). Furthermore, compared to baseline, a reduction in TNF- α was observed in both atorvastatin treatment group (atorvastatin 10 mg: 0.945 ± 0.588 pg/ml vs. 1.145 ± 0.622 pg/ml, $p < 0.05$ and atorvastatin 40mg: 0.993 ± 0.627 pg/ml vs. 1.139 ± 0.620 pg/ml, $p = 0.085$). The changes in FMD and Alx were not correlated with the decrease of LDL cholesterol or BNP serum levels in either atorvastatin treatment groups.

Conclusion: Short term treatment with high dose atorvastatin improves arterial wall properties and inflammatory status in ischemic HF patients irrespectively of LDL and BNP reduction. These results favor pleiotropic effects of atorvastatin in patients with heart failure.